Volume 75

Number 12

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DECEMBER 1958

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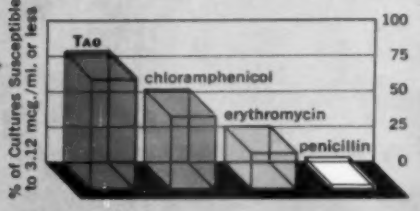

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References: 1. English, A. R., and Fink, F. C.: Antibiotics & Chemother. (Aug.) 1958. 2. English, A. R., and McBride, T. J.: Antibiotics & Chemother. (Aug.) 1958. 3. Wennersten, J. R.: Antibiotic Med. & Clin. Therapy (Aug.) 1958. 4. Celmer, W. D., et al.: Antibiotics Annual 1957-1958, New York, Medical Encyclopedia, Inc., 1958, p. 476.

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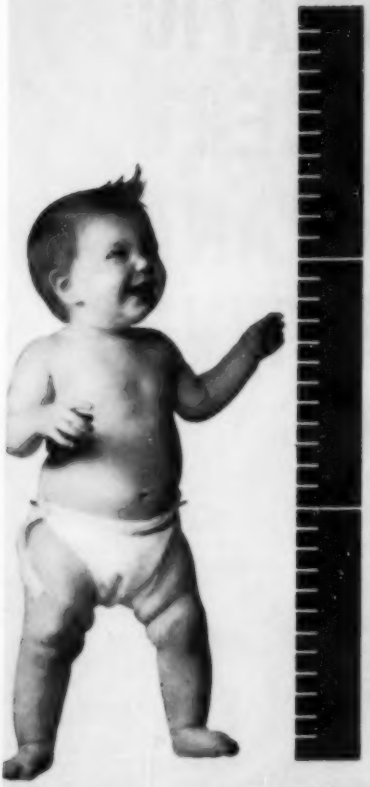

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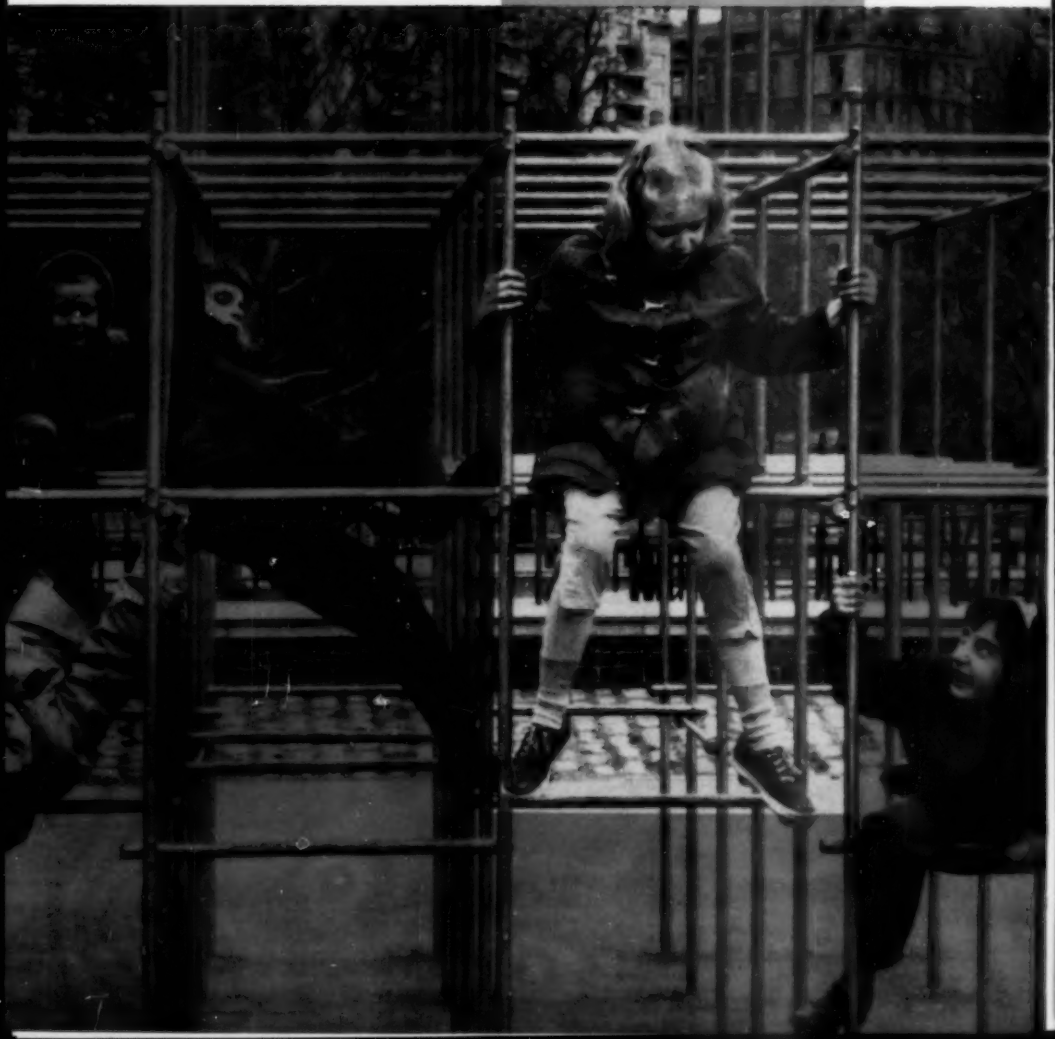

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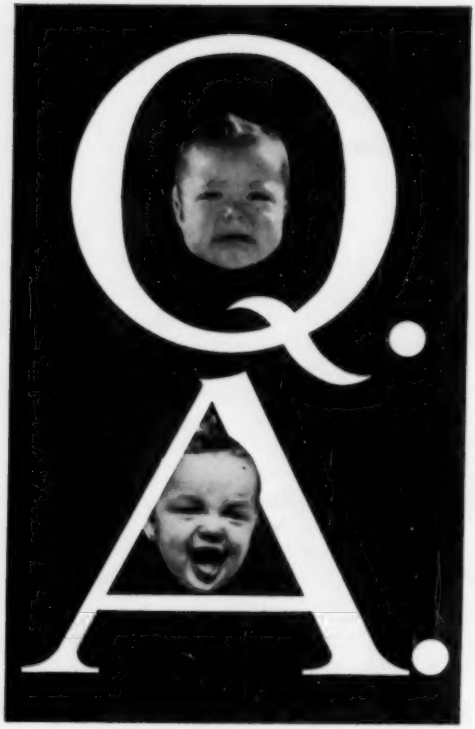
Cover - Clemens von Pirquet (1874-1929)

Clemens von Pirquet was professor of diseases of children at the Universities of Breslau and Vienna, and professor of pediatrics at Johns Hopkins University, Baltimore. He was generally known for the cutaneous tuberculin test that bears his name. Through his studies of serum sickness and stages of vaccination, he discovered the principle of allergy. As a result of his continuing studies and discoveries, Dr. von Pirquet introduced and contributed new concepts and systems in medicine that threw light on the pathogenesis of asthma and eczema. His research on mortality statistics extended his work on allergy to include changes in the reactivity of the organisms at different age periods. He also described a new system of feeding known as the "Nem system," in which he used the sitting height instead of the weight, as an index of food requirement.

(*"Pioneers of Pediatrics", by Abraham Levinson, M.D.*)



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News and Notes

The American College of Allergists, Boulder, Colorado, announces March 15-30, 1959 as the dates for their Graduate Instructional Course and Annual Congress to be held at the Mark Hopkins Hotel, San Francisco, California. John D. Gillaspie, M.D., Treasurer, can be contacted at 2049 Broadway, Boulder, Colorado, by those interested.

•

The Sister Elizabeth Kenny Foundation announces continuation of its post-doctoral scholarships program for promoting work in the neuromuscular diseases field. Candidates from U. S. and Canadian medical schools are eligible. Inquiries should be addressed to Dr. E. J. Huenekens, Medical Director, Sister Elizabeth Kenny Foundation, Inc., 2400 Foshay Tower, Minneapolis 2, Minn.

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The University of Washington, School of Medicine, Department of Pediatrics, announces that it is setting up a research training program with the collaboration of members of the staff of the Departments of Biochemistry Pathology, Anatomy and Medicine. The program is limited to graduates with the M.D. degree, interested in full-time academic careers in research and teaching fields. Those interested can write to: Program Director, Bruce Mackler, M.D., Dept. of Pediatrics, University of Washington, School of Medicine, Seattle, Washington.

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FUNNEL CHEST

The Status 360 Years After Its First Description

Charles W. Lester, M.D.*

New York, N. Y.

In 1596 Bauhinus[1] observed a funnel chest deformity in a boy. He wrote a remarkably good description of the condition which was published in 1600. Since that time other accounts of the deformity have appeared sporadically in the literature so that in 1936 Ochsner and DeBakey[2] were able to list 142 such articles in a paper in which they described a case. These dealt with a limited number of cases and it was not until Brown[3] established the fundamental pathogenesis of the condition and laid down the principles for its surgical correction that larger series of cases appeared in the literature. However, there is still confusion about the condition due to the fact that the deformity is relatively uncommon, no really large series of cases have been reported, and many of the series deal only with cases in a limited age group. The series on which this paper is based comprises 156 operative cases ranging in age from 3 months to 40 years, an undetermined number of cases which, for various reasons, did not come to operation, and a considerable number of cases of anterior chest wall deformity confused with funnel chest but where protrusion was the prominent feature. These cases were seen in the Chest Deformity Clinic of the Hospital for Special Surgery in New York and in private practice. It is not a large series but there are enough cases to permit some conclusions to be drawn.

Funnel chest is a funnel-shaped depression of the anterior chest wall which is deepest in the region of the xiphoid. (Fig. 1) The xiphoid lies in a plane posterior to the second sternal segment and, in infants and children, moves backward during inspiration in a paradoxical manner. It must not be confused with the conical depression which accompanies arcuate protrusion of the sternum nor with the pardoxical respiration associated with dyspnea in an infant or young child. In the first instance the xiphoid does not have a paradoxical respiratory motion. In the second instance relief of the dyspnea will stop the paradoxical respiration which is due to the sudden build-up of negative intrathoracic pressure during forced inspiration in the labile thorax of infancy.

*Consulting Thoracic Surgeon, Hospital for Special Surgery, and Roosevelt Hospital, New York. Consulting Surgeon, Bellevue Hospital, New York

The funnel-shaped depression of the anterior chest wall is produced by the backward curvature of the sternum starting at the junction of the manubrium and gladiolus and progressing so that the xiphoid approaches the vertebral column. As the sternum curves backward the costal cartilages follow it and curve in the same direction. This diminishes the antero-posterior measurements of the thorax, causes the involved ribs to slant caudad, and displaces

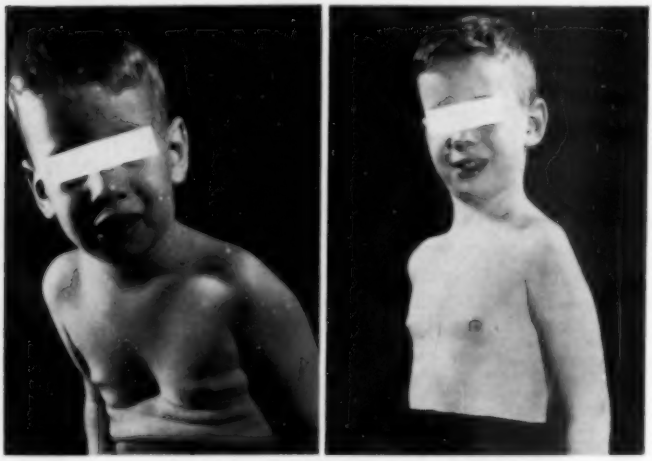


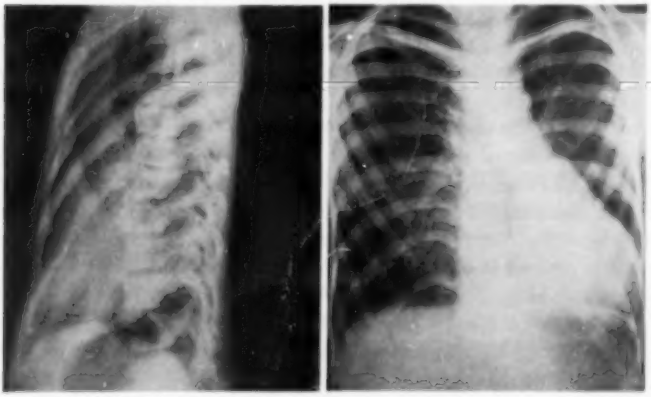
Fig. 1 Fig. 1 A

Fig. 1. Boy, 2½ years old; funnel chest noted at birth; repeated attacks of bronchitis and pneumonitis beginning at age of 15 months. Note round back posture in addition to funnel chest.
Fig. 1 A. Child shown in Figure 1 two years after radical sternoplasty. Note erect posture, normal development and absence of funnel chest.

the heart into the left hemithorax. (Fig. 2) In all cases there is a transverse groove across the chest which resembles the Harrison's groove of rickets and the abdomen seems to protrude so as to give a potbelly appearence. Round back and a mild degree of scoliosis are often associated with the deformity. In infants and young children the funnel chest is usually symmetrical but in some cases asymmetry develops as the child becomes adolescent. When this occurs the more prominent part of the chest is apt to be on the left although it can be on the right. If the patient is a girl the breast on the deeper side will not develop as well as the other. Occasionally, other elements of a protrusion deformity, especially the arcuate type (pigeon breast), occur to further confuse the picture.

Heredity plays an important part in the etiology of this deformity but the characteristic is often recessive and the responsible forebear may be impossible to find or is recognizable only by discovering the deformity in a relative in a collateral branch. On the other hand it is not rare to find the deformity in three or four successive generations or in siblings.

Here it must be noted that the forebear may have a pigeon breast



(c) *Journal of Thoracic Surgery*

Fig. 2. Antero-posterior and lateral roentgenograms of an extreme case of funnel chest in a 7-year-old girl. Note backward curvature of sternum, caudad slope of anterior ribs and cardiac displacement. Straight spine and slight potbelly suggest Marfan's syndrome not borne out by subsequent development.

(pectus carinatum) and not a funnel chest (pectus excavatum). The reverse is also true. Furthermore in siblings one may have a funnel chest and another a pigeon breast. In Marfan's syndrome pectus excavatum is about as frequent as pectus carinatum, at least in my experience. This, together with the fact that elements of both types of deformity may be found in the same individual leads me to believe that both deformities should be classified together. This concept has been discussed in a previous paper.[4]

Two factors are responsible for the pathogenesis, viz: the pull of the diaphragm and the disproportionate growth of the ribs. Of these, the pull of the diaphragm is the most important and is active in all cases before maturity. The disproportionate growth of the ribs is a potent factor in those cases which appear several years after birth, espcially as the child approaches puberty.

My own observations bear out the theory that the diaphragmatic

pull is due to a congenitally short central tendon of the diaphragm which causes the diaphragm to draw the xiphoid backward during inspiration. There is no other muscle which can produce this action. As the xiphoid moves backward while the lower ribs move forward a groove is made across the chest which resembles the Harrison's groove of rickets. The same action also gives the appearance of potbelly. Actually the abdomen does not protrude; the chest recedes Because of this short central tendon the diaphragmatic action is definitely comprised, a situation which is responsible for some of the cardinal symptoms of the deformity.

Disproportionate rib growth, especially in the lower ribs, seldom becomes apparent until the age of five and often not until the increased growth of puberty takes place. There is probably a moderate shortening of the central tendon which is of minor importance until the ribs begin to accelerate their growth as puberty approaches. Then the restraining action of the diaphragm plus the rib growth produces the funnel chest deformity. This resembles the deformity that is present at birth except that the anterior portions of the ribs do not slant as much in a caudad direction and the antro-posterior diameters of the chest are not as much decreased. The symptoms are the same although they do not develop as early. If the ribs grow more on one side than the other an asymmetrical deformity will be produced.

The effects of funnel chest are definite and can be very severe. They involve the lungs and the heart and, in general, vary in proportion to the degree of deformity, although this is not always the case. When investigating symptoms, it must be remembered that in most instances the patient has never known what it is to be normal and hence cannot judge his own departure from the normal. He must be compared with normals in his own age group.

Lung volumes are apt to be diminished, sometimes to a considerable extent but seldom enough to produce important symptoms. Most of the respiratory symptoms are produced by the restricted diaphragmatic action which interferes with an effectual cough. A minor respiratory infection, easily handled by a normal child, can go on into bronchitis or pneumonitis because the patient is unable to cough out his secretions. (Fig. 3) In infants this has produced fatal results and is the most important indication for operation in a child less than a year old. In the older group, repeated acute respiratory infections frequently go on to any of the chronic respiratory infections which, in turn, can bring about emphysema and cor pulmonale in later life, a condition which is not of immediate concern

to the pediatrician. In a great many cases there is a functional systolic heart murmur which may be difficult to differentiate from an organic murmur.[5] It has no other significance. The real cardiac symptoms are dut to restriction of heart action by pressure of the sternum or of the bony thorax because the sternum has so displaced the heart. The condition closely resembles constrictive pericarditis and may be severe enough to keep the patient in bed or so mild that it only limits prolonged, strenuous activity. In general, this is noticed because the child tires easily, avoids strenuous games and is unwilling to exert himself except for short periods. If the heart does not shift to the left as the sternum encroaches on it a slight depression can cause marked symptoms.

The systoms described are physiological in nature but the psychological effects of funnel chest are just as important and often more severe. It is unusual to find an individual over the age of 6 years with a definite funnel chest deformity who is not conscious of the deformity and embarassed by it. For that reason the victim avoids bathing beaches, refuses to participate in athletics and, if a girl, shuns social events that require conventional evening attire.

Cardio-pulmonary functional tests to demonstrate these symptoms are very unsatisfactory. It has already been mentioned that lung volumes are reduced, sometimes as much as 25% or more, and that this amount is not enough to make any great difference in the activity of the patient. In testing heart function Wachel, Ravich, and Grishman[6] have shown that in cases with considerable deformity the cardiac output decreases with vigorous exercise. I think this is so in most cases with obvious deformity. However, to subject a patient to prolonged, exhausting exercise with a cardiac catheter in the ventricle is a hazardous procedure and, at present, there is no other accurate means of measuring cardiac output under these conditions. It is, therefore, unjustifiable to state that the increased exercise tolerance which follows operation for funnel chest is psychological. Rather, it should be said that there is no laboratory method now available to demonstate the improvement. Certainly, the great majority of cases of funnel chest deformity show a decided physiological improvement as well as psychological improvement after the deformity has been surgically corrected.

The treatment of funnel chest is surgical but because the condition is so complex the optimum age for operation varies with the individual. If repeated severe respiratory infections occur it can be done as early as the age of three months (Fig. 3), or before the age of one year if the deformity is bad and seems to be getting worse.

Under these circumstances a simple sternoplasty can be performed to tide the baby over until there has been a change for growth and development. A radical sternoplasty may be necessary a few years later but the baby has had an opportunity to develop in the meanwhile and there is better than an even chance that no further surgery will be required. Radical sternoplasty can be performed in infants but, if symptoms do not demand, it is better to wait until the child is two years old or over because he can then tolerate the procedure better and there is less liklihood of subglottic edema from the use of the intratracheal anesthetic tube. In any case, a well-developed deformity present in infancy should be corrected by surgery before the age of five years because after this time uncorrectable changes may take place.

However, if the deformity is slight or develops late, operation should not be undertaken until there has been a chance to grow because growth may change the situation one way or another. In any case, operation should be done for every obvious deformity before the age of puberty, particularly if there is a tendency to an asymmetrical deformity. Of course, if the optimum time is missed radical sternoplasty can be done at anytime but the correction will not be as good.

There are two types of operation. One is a simple procedure in which the seventh cartilages are resected subperichondrially; the xiphoid is detached from the sternum and the mediastinum entered; and the beds of the seventh ribs, and the sixth ribs if possible, are detached from the sternum with the xiphoid. The resiliency of the ribs and sternum is relied upon to correct the sternal curvature and no attempt is made to elevate the sternum. This operation should rarely be undertaken after the age of one year. At best, as mentioned before, the recurrence rate is too high but it can be a life saving procedure and should not be discarded even though it is expected that a radical operation will have to follow in a year or two. The operation can be quickly done, endotracheal anesthesia is not essential and the patient can easily tolerate it.

The radical procedure requires the bilateral superichondrial resection of the third to the seventh costal cartilages (10 ribs) from the sternum laterally to the point where the cartilage begins to turn backward. A transverse wedge osteotomy is made across the second sternal segment down to the posterior table; the xiphoid is detached from the sternum and the ventral mediastinum entered; the sternum is then elevated to a normal position by breaking it up along the wedge osteotomy. The periosteum is sutured across the osteotomy

and the position further maintained by passing two wires transversely behind the sternum and carrying the ends out onto the anterior chest wall where they are twisted into a transverse support of duralumin. This can be removed in a week with no more discomfort to the patient than the removal of stitches. Some surgeons[7] dispense with the wires and support but, in my hands, this can be very unsatisfactory, especially since postoperative cough is common and subglottic edema with attendant dyspnea is not unusual, either of which can compromise the operation if the sternum is not supported.

Fig. 3. Infant with marked funnel chest; persistent cough after two attacks of pneumonitis accompanied by cyanosis and high fever; simple sternoplasty at age of three months with complete relief of pulmonary symptoms.

The patient can get out of bed the day after the operation and is usually ready to go home a week later.

In children past the age of puberty a rib strut from the patient or from the bone bank can be passed transversely under the sternum and fastened to the ends of the resected cartilages. No external support is needed and there is nothing to remove. It is chiefly valuable in girls whose breasts get in the way of external support.

Because the perichondrium is left, the cartilage regenerates promptly and the chest wall is stiff enough at the end of a week to prevent paradoxical movement with ordinary respiration. In some cases the regenerated cartilage has been replaced by normal bone.

The mortality rate of less than 1% is very low considering the magnitude of the radical procedure and the results are generally excellent. (Fig. 1 A) There is often a slight residual depression but I have had to reoperate upon only two cases for recurrence after

the radical operation. In three other cases there has developed a depression as the child continues to grow but this has not been bad and, since the xiphoid moves in a normal manner, the depression is not a true recurrence and does not produce symptoms.

The simple sternoplasty has a higher percentage of recurrence, as has been mentioned. When done for the proper indications the operation is not in the class of elective procedures and a 40% recurrence rate is not too high a price to pay for the normal development of an infant for the first two years of life. Radical sternoplasty can always be performed later, something which might not have been possible without the simple preliminary operation.

SUMMARY

Funnel chest (pectus excavatum) is a deformity of the sternum and costal cartilages characterized by a funnel-shaped depression maximum in the region of the xiphoid. The xiphoid lies in a plane posterior to the manubrium and, in infants and children, retracts during inspiration.

The deformity is usually present at birth or develops shortly thereafter although an appreciable number do not appear until the child has had a chance to grow, particularly just before puberty.

Heredity is an important factor in the etiology.

The pathogenesis can be explained by a short central tendon of the diaphragm in all cases and over-growth of the ribs, in addition, when the deformity develops in childhood.

The symtoms are respiratory, because of an ineffectual cough as a result of the deformed diaphragm; cardiac, due to pressure on the heart by sternum and ribs; and psychological, due to the appearance of the deformity.

The treatment is surgical; the risk is slight; and the results are good.

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THE SERUM INORGANIC PHOSPHOROUS CONTENT OF NORMAL EGYPTIAN INFANTS AND CHILDREN

S. Awwaad, D.CH., M.D.*

and

E. A. Eisa, M.SC., PH.D.*

Cairo, Egypt

Phosphorous is among the most important inorganic constituents of the blood. Most authorities have found that serum inorganic phosphorous in children ranges between 4 MGM% and 6 MGM%, that is, it is roughly double that of adults. However, what the phosphorous changes are and when they occur as the newly born infant passes through its infancy, childhood and adolescence is not generally agreed upon. It is the purpose, therefore, to determine in this review the normal serum inorganic phosphorous and its physiological variations in Egyptian infants and children as they grow from birth to adolescence.

REVIEW OF THE LITERATURE

Prior to 1920, the knowledge on blood inorganic phosphorous was meager. McKellips, DeYoung and Bloor[1] (1921) obtained a range of 1.2 mgms. to 4.6 mgms. for infants from birth to two weeks of age. Plass and Tompkins[2] (1923) found comparatively low values for inorganic phosphorous of newborn infants, (3-5 mgms./100 cc.). Howland and Kramer[3] (1921) in a series on infants and children, between three months and three years of age, found an average of 5.4 mgms./100 cc. They[4] also found (1921) that all children with less than 3 mgms. of phosphorous per 100 cc. of blood showed evidence of rickets. William[5] (1928) found that the difference in age from 1 year to 11 years made no difference in inorganic blood phosphorous. His work, however, was not limited to normal children.

Hess and Lundagen[6] (1922) showed that there is a slight seasonal variation in the inorganic phosphorous of the blood of children. They considered this a physiological phenomenon. The highest level of the tide occurred in June and July, then it diminished to touch the lowest level in March. A year before, they[7] pointed out the marked similarity between the seasonal phosphorous curve and the seasonal incidence of clinical rickets. Riesenfeld[8]

* From the Pediatric and Chemistry Departments, Ein Shams University, Cairo, Egypt

(1925) found that the blood phosphorous of the mother is more constant, showing no seasonal variation as in the child and stated that this favors the theory that seasonal variation is confined to rachitic or pre-rachitic cases, or occurs with seasonal variation in growth. Bullock[9] (1930) found that the average for the newly-born infant is less than for any period under five years, he found that the level rises from birth to reach a peak at seven months of age, then gradually decreases to 19 years of age, when a more sudden drop occurs. Stearns and Warweg[10] (1933) also found comparatively low values in the newly-born, then the values steadily rise to 4 months of age, or 6 months of age, and become nearly constant between one and twelve years, with only a slight decrease from 5.5 to 5.0 mgms. In this respect, their values compare well with those of Bullock's, but are definitely greater than the average of 3.9 mgms. reported by Federova[11] for Russian children and by Sokolovitch[12] for children in Glasgow. In the Stearns and Warweg[10] study, the drop to the adult level occurred at about 13 years of age, that is, much earlier than in Bullock's[9] or in Tisdall and Harris[13] works. Awatif and Abul Fadl[14] (1954) found a tendency for high levels of serum inorganic phosphate in the newly-born Egyptian infants. In 51 percent of their cases, the values exceeded 6 mgms./100cc. which is the maximum figure recorded for children. They noticed also that most of the high figures are in infants in their first week of life.

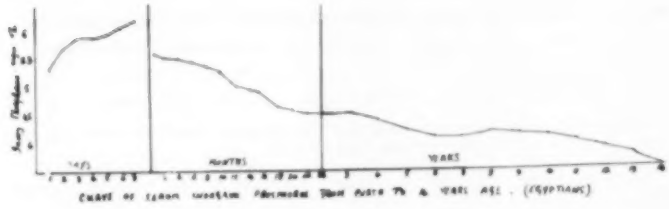
EXPERIMENTAL WORK

One hundred and fourteen cases were studied of which thirty were newborns, a similar number representing the period of infancy, while the rest included the age period from two to fourteen years. The cases were selected from healthy newborns, infants and children who accompanied their ailing relatives in the out-patient of Abbassa Children's Hospital. The cases were thoroughly examined clinically to exclude any abnormality in their general health. From each case, 2.5 cc. of blood was taken for the estimation of serum inorganic phosphorous content using the method adapted by Fiske and Subba Raw[15] in which the proteins of the blood are precipitated with trichloroacetic acid. The protein-free filtrate is treated with acid molybdate solution which forms phosphomolybdic acid from any phosphorous present. The phosphomolybdic acid is reduced by the addition of 1, 2, 4 aminonaphthol sulfonic acid reagent to produce a blue color whose intensity is proportional to the amount of phosphate present.

TABLE I

The results are given in the following tables and graph, and show the age and sex of the cases studied and their serum inorganic phosphate levels.

Average age		No. of cases M	No. of cases F	No. of cases	Average Serum Inorg. Phosph. Mgm.%.
1	day	2	–	2	5.33
2	days	1	1	2	5.8
3	"	1	1	2	5.8
4	"	2	4	6	5.7
5	"	1	2	3	5.9
6	"	1	1	2	6.1
7	"	2	–	2	6.2
10-29	"	6	5	11	5.7
1- 3	months	4	2	6	5.6
4- 7	"	3	3	6	5.5
8-11	"	3	1	4	5.3
12-15	"	3	3	6	4.9
16-20	"	2	2	4	4.7
21-24	"	2	2	4	4.5
2- 3	years	5	2	7	4.5
3- 4	"	2	4	6	4.4
5- 6	"	6	4	10	4.3
6- 7	"	1	5	6	3.9
7- 8	"	1	3	4	4.1
8- 9	"	3	2	5	4.1
9-11	"	4	3	7	4.1
11-14	"	4	5	9	3.7
		59	53	114	



DISCUSSION AND COMMENTS

(a) *In the neonatal period:* Serum inorganic phosphorous shows the highest levels. This is more pronounced in the first week where the values in eleven out of nineteen cases studied exceeded the maximum figure recorded for children (6 mgms/100cc.). In this respect, our results are in agreement with those of Awatif and Abul Fadle[14] (1954), but they differ from those of McKellips et al[1] (1921). Plass and Tompkins[2] (1923), Bullock[9] (1930) as well as Stearns and Warweg[10] (1933) who found that the levels in the neonatal period are lower than for the first five years of childhood.

However, the figure of the latter authors is in fair agreement with ours, as illustrated in Table II.

TABLE II

Comparative study of the values of the S. I. P. levels in the neonatal period as found by different authors.

Authors	No. of cases	Serum Inorg. Phosphorous mgm. %
McKellip, et al[11] (1921)	21	3.0
Plass and Tompkins[2] (1923)	11	4.5
Bullock[9], New Orleans (1930)	13	4.82
Stearns and Warweg[10], Iowa City (1933)	18	5.6
Awatif and Abul Fadl[12], Egypt (1954	109	5.6
Awwaad & Eissa, (present series), Egypt (1957)	19	5.9

The increased serum inorganic phosphorous found may be explained by the great requirement of phosphorus to accommodate the highly active anabolic and catabolic processes that take place during that period.

(b) *During the first two years of age:* The cases show a relatively high level during the period from one to six months with a high average of about 5.5 mg.%. This is followed by a relatively rapid drop to the end of the second year when it becomes 4.5 mg.%. In spite of this drop, it is still at a higher level than that of the rest of the childhood period. These findings are comparable to those of Bullock[9] (1930) and those of Stearns and Warweg[10] (1933) in the first six months period as shown in Table III. This raised level may contribute to the rapid growth that occurs during that time.

TABLE III

Comparative study of serum inorganic phosphorous levels as found by different authors in the first six months.

Authors	No. of cases	Age Period	Average Serum Inorg. Phos. content
Jones and Nye[16] (1921)	4	1-7 months	3.0 mg. %
Bullock[9] J. K. (1930)	23	1-6 "	5.5 " "
Stearns and Warweg[10] (1933)	15	1-6 "	5.8 " "
Awwaad & Eissa (1957) (present series)	10	1-7 "	5.5 " "

(c) The cases studied were fifty-four. The average figures obtained, as shown in the graph, show little variation with a slight drop from 4.5 at 2 years to 3.5 mg/% at 14 years with an average of 4.2 mg.%. This slow and slight variation is in agreement with the previous studies done in that field. However, the figures obtained in our cases fall between the high values reported by Stearns and Warweg[10] (1933) and Bullock[9] (1930) and the relatively low ones reported by Fedorova[15] (1930) and Sokolowitch[16] (1931) as shown in Table IV. It may also be noticed that in the present series

the adult levels have almost been reached by 14 years of age, without showing any abrupt drop as that mentioned by others. Bullock[9] and Tisdall and Harris[13] in their study found the adult level was reached at about the same age but with an abrupt drop. In Stearns and Warweg[10] series it was reached at about 19 years of age and also with an abrupt drop.

TABLE IV

Comparative study of the average serum inorganic phosphorous levels in children of different nationalities.

Authors	No. of cases	Nationality	Age Period	Aver. Serum Inorg. Phos. Content
Jones and Nye[16] (1921)	30	Amer.	3-13 yrs.	2.9 mg. %
Bullock, J. K.[9] (1930)	130	"	1-19 "	4.8 " "
Stearns and Warweg[10] (1933)	55	"	1-12 "	5.2 " "
Fedorova[11] (1931)		Russ.	1-12 "	3.9 " "
Sokolovitch[12] (1931)	12	Brit.	4-12 "	3.9 " "
Awwaad & Eissa (1957) (present series)	54	Egypt.	2-14 "	4.2 " "

SUMMARY AND CONCLUSIONS

1. A review of the literature on serum inorganic phosphorous in infants and children is given.
2. This work represents the first study of serum inorganic phosphorous content among normal Egyptian infants and children.
3. A total number of 114 cases were studied.
4. The variations that occur from one age period to the other are demonstrated.
5. The results obtained in the present series were discussed and compared with similar ones reported in other countries.

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COLLOIDAL EMOLLIENT BATHS IN PEDIATRIC DERMATOSES

LIONEL A. DICK, M.D.*

New York

Skin problems of infants and children, according to several recent reports, constitute approximately ten percent of cases seen in private and institutional practice. Many of the cutaneous diseases seen in this age group are also encountered in older age groups, but the management of skin conditions in infants and children present special difficulties because of numerous anatomical and physiological differences.

The skin of the child, and especially the infant, differs in many respects from that of the adult. Anatomically it is thinner, less cornified and less hairy. The average skin thickness of the adult is 2 mm. At birth, the infant's skin averages around 1 mm., and remains not more than 1 mm. through childhood.[1] Physiologically, the sweat and sebaceous gland secretions are less active. Physically, the skin is more permeable to fluids, and immunologically, the child's skin is inferior to that of the adult.

In infancy and childhood as in old age, the skin tends to be on the dry side. This period coincides with decreased gonadal activity, which influences the pilo-sebaceous mechanism.

Although soaps and the newer "non-soap" detergents are satisfactory cleansing agents for physiological skin, their use is not desirable where symptoms such as inflammation, irritation or pruritus are present. It is thought that soaps and detergents cleanse because of their ability to lower the surface tension of water, thus permitting more intimate contact between the water and the material to be cleansed (the skin). These materials are, therefore, classed as surface active agents. Thus, they enable the water to penetrate between the skin and the particles of soil. The soap or detergent forms an emulsion of the fats, greases and water on the surface of the skin. The insoluble particles of dirt are then wetted, surrounded and suspended. Mechanical action also aids in loosening the dirt from the surface. Finally, the solution containing the emulsified lipids and suspended dirt is rinsed away.

It is obvious that the use of irritating soaps and the new deter-

* *Assistant in Dermatology, Skin and Cancer Unit, N. Y. University-Bellevue Medical Center*
Assistant Adjunct, Beth Israel Hospital

gents should be reduced or eliminated in certain skin conditions[2], inasmuch as these dermatoses present features which are similar to those which occur from the excess use of soap. Among these conditions are congenital ichthyosis, the dry avitaminosis A, and endocrine dysfunction (hyperthyroidism). There are also skin conditions in which clinical empiricism suggests the avoidance of soaps and detergents. This group includes the acute and weeping eczematous dermatoses (contact dermatitis, diaper rashes, atopic dermatitis, seborrheic dermatitis, fungous infections) which are notoriously sensitive to soaps. Often the mere avoidance of soap contributes to favorable results.

Oatmeal has been used for many years for its cleansing and therapeutic properties. It is interesting to note that despite the relative lack of information on the cleansing mechanism of oatmeal, it continues to remain a widely-used agent in dermatologic therapy as a soothing and cleansing agent. Glaser contends[3] that inflamed eczematous skin acts as a semi-permeable membrane and, thereby, permits relatively large quantities of bath water(which is hypotonic) to enter. He proceeds further to state that the addition to the bath water of colloidal materials such as oatmeal, decreases and controls the osmotic pressure of the water with respect to the skin.

In recent years, the concentrated starch-protein complex of oatmeal*[4, 7] has been made available. Inasmuch as this is the colloid-producing fraction of the oat grain, colloidal oatmeal has superior demulcent and cleansing properties when compared with regular oatmeal or starch. More recently, colloidal oatmeal has been made available in a super-oiled form** in which the colloidal oatmeal has been impregnated with 35% liquid oils for additional emolliency and skin lubrication.

This new "Oilated" colloidal oatmeal appears to have wide usage in many pediatric dermatoses since it combined the recognized soothing and cleansing qualities of colloidal oatmeal with the emolliency of bland oils.

The principle of colloidal emollient baths appears to be in accord with Blank's concept[5] that water, in combination with an occlusive film, such as oil, is the most effective plasticizer of keratinized or dry skin. In this bathing procedure, some of the bath water is imbibed by the skin tissues thereby softening the skin. The water which has softened the skin is then retained by means of the thin

*Aveeno®.
**Aveeno® "Oilated".

occlusive film of oil which remains on the skin. In other words, the thin film of oil acts as an effective barrier by retarding the evaporation of water from the skin surface thus helping to keep the stratum corneum hydrated and flexible until it receives moisture from the underlying tissues.

METHOD

In order to determine the effectiveness of these colloidal emollient baths as adjunctive therapy, it was decided to conduct a study on a series of pediatric patients manifesting various dermatoses associated with dryness of the skin. Where indicated, other specific medicaments such as corticosteroid and antibiotic preparations, were used.

(TABLE I)

Diagnosis	Number of Patients
1. Atopic dermatitis	70
2. Contact dermatitis (including intertrigo and diaper rashes)	30
3. Fungous infections (moniliasis)	20
4. Seborrheic dermatitis	18
5. Miliaria (prickly heat)	2
6. Ichthyosis	4
7. Drug eruptions	8
	152

COMMENT

In all cases reported, baths of colloidal oatmeal in a super oil form proved to be excellent adjuncts to the therapy used. They exhibited a remarkable lack of irritating properties and, at the same time, showed excellent soothing qualities as well as an effective cleansing ability. The parents were unanimous in finding the colloidal emollient bath easy and convenient to use. In several cases, where the child manifested a distinct tendency to dryness, the baths were adopted as a routine cleansing and protective measure even after the dermatoses had completely subsided.

CONCLUSION

"Oilated" colloidal oatmeal baths provide the pediatrician with a non-irritating, soothing, cleansing and protective agent which is well accepted by both parent and child, for prolonged use.

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Clinical Conference

ST. LUKE'S HOSPITAL, New York
September 9, 1958

Dr. J. Frederick Eagle, Jr.
Director of Pediatrics, presiding

CASE PRESENTATION

DR. EAGLE: Today we will present a child who remains a diagnostic problem. There is some evidence to suggest glycogen storage disease and other evidence to suggest an unusual form of liver disease.

DR. ELEANOR T. ROTH: Pediatric Intern. The patient is a three year old Puerto Rican male with a chief complaint of diarrhea for 2½ years with an exacerbation occurring three days prior to admission.

His mother had had an uneventful pregnancy, and delivery was spontaneous at term. The birth weight was 6 lbs. 7 oz. The patient was discharged from the hospital as healthy. Shortly after discharge he was noted to have green skin. At approximately one month of age he was hospitalized for one month. Subsequently he did poorly with vomiting and diarrhea occurring about two or three times a day, until one year of age when green skin was again noted and the patient was rehospitalized, this time for one year. During that year's hospitalization there was noticed progressive abdominal enlargement, and after discharge he continued to do poorly.

The child came to the United States seven months prior to the present admission. The diarrhea persisted and retarded growth and development was noted. The child was unable to walk alone. He first stood without support at the age of 24 months. His speech was limited to "Mamma" and "Papa".

Three days prior to admission he developed loose stools three or four times a day unassociated with fever, anorexia, nausea, or vomiting.

Physical examination revealed a poorly developed, obviously physically retarded, alert, Puerto Rican male unable to walk alone.

The liver was three finger breadths below the right costal margin. There was no splenomegaly. No icterus was noted. His height of 28" was eight standard deviations below the mean. His weight was 17 lbs., 15 oz., four standard deviations below the mean. The head circumference was 1.4 standard deviations below the mean.

His hemoglobin on admission was 7.2 grams. The serum iron was low and the iron binding capacity high-normal. This anemia was treated with ferrous-sulfate and the hemoglobin subsequently rose to 11.3 grams. Liver studies showed a total serum protein of 7 grams %, 4.7 grams % of albumin, 2.3 grams % of globulin. Total cholesterol was 143 mg.%, cholesterol esters were 119 mg.%. Serum bilirubin was 0.1 mg.%, glutamic oxalacetic transaminase was 26 units, zinc turbidity 7.2 units, B.S.P. 4% retention in 15 minutes, 2% retention in 45 minutes. The Hanger test was negative. Alkaline phosphatase was 17 King-Armstrong units. Galactosemia was ruled out by the absence of galactose in the urine.

X-ray of the skeleton showed a bone age of one year. Protein Bound Iodine was normal, 5.1 micrograms %. Radioactive iodine uptake was normal. Barium enema failed to reveal any abnormalities of the gastrointestinal tract. Sweat test was normal. Stool was negative for intracellular fat. His glucose tolerance curve was normal, but an epinephrine test showed a low curve, with an increase in the blood sugar of 30 mg.% as compared to a normal response where the glucose rises 50-60 mg.%. The response to glucagon was similar, with only a modest rise in the blood glucose. The only time we have been able to demonstrate a low blood sugar was after a 36 hours fast when it fell to 46. At this time he had 4+ acetonuria.

Liver biopsy was done two days ago. Grossly the liver was enlarged and showed a flaccid edge with what appeared to be some areas of regeneration. There were beginning areas of fibrosis scattered throughout the liver. Microscopic frozen section showed normal lobular configuration of the liver. The only pathological finding so far has been areas of fibrosis.

DR. EAGLE: Certain tests suggested a diagnosis of glycogen storage disease and I have asked Dr. Van Itallie to discuss this aspect of the problem.

DR. THEODORE B. VAN ITALLIE: Director of Medicine, St. Luke's Hospital. First, let us review some of the information that is available on the biochemistry of glycogenesis and glycogenolysis in the liver. Particular reference will be made to the role of the

various enzymes that are concerned in these processes, and the hormones that influence the rate at which these enzymatic transformations take place.

In Figure 1 we see that glucose, under the influence of glucokinase is phosphorylated to glucose-6-phosphate which, under the influence of phosphoglucomutase is transformed to glucose-1-phosphate. This substance in turn is incorporated into glycogen under the influence of phosphorylase and the branching enzyme.

Fig. 1. Enzymatic reactions involved in glycogen synthesis and degradation in liver.

The other hexose sugars in the diet that enter the portal vein, such as fructose and galactose, are converted by a series of enzymatic steps to glucose-1-phosphate which, in turn, incorporates into glycogen. Certain three-carbon compounds, such as the glycogenic amino acid, alanine, and pyruvate, also can be converted readily into glycogen.

When glycogen is converted to glucose by the process of glycogenolysis, these reactions are reversed. Glycogen, again with the help of phosphorylase, and in the presence of the debranching enzyme (the brancher and debrancher are not two-way enzymes, they only work in one direction) is transformed to glucose-1-phosphate in the presence of inorganic phosphate. The glucose-1-phosphate is changed by phosphoglucomutase to glucose-6-phosphate,

and then in the presence of the enzyme glucose-6-phosphates, the glucose-6-phosphate is changed to glucose. In this form it can diffuse into the circulation via the hepatic venous blood. All these reactions up to free glucose formation take place within the liver cell, and only then can the glucose leave the cell.

Apparently inorganic phosphate remains in the liver, according to studies we have done on phosphate gradients across the liver during glycogenolysis. If the transformation of glycogen to glucose is blocked (as occurs in glycogen storage disease) it can be seen that, in theory, there are a number of enzymatic points at which such a blockade could occur.

In recent years, it has become possible to assay the livers of patients with glycogen storage disease for several of the enzymes concerned with glycogenolysis and also to examine the structure of glycogen to determine whether different kinds of glycogen are present, and if so, how they differ.

By examining our diagram of glycogenolysis, we can see that if there were a deficiency of glucose-6-phosphatase, glucose would not be able to escape into the circulation. If there were a deficiency of phosphoglucomutase, glycogen could not be formed or once formed, would not be able to change to anything beyond glucose-1-phosphate. In the absence of phosphorylase, or in the absence of debrancher or brancher, there might be either failure to form glycogen, or failure to release it, or the formation of abnormal glycogen, depending upon the enzyme involved.

DR. EAGLE: In going from some of these other sugars, particularly galactose, can you go from galactose to glycogen without phosphorylation?

DR. VAN ITALLIE: No, all the dietary hexoses must be phosphorylated in order to be further metabolized. However, I might add that insulin may facilitate the phosphorylation of glucose by glucokinase in the liver but it does not seem to affect any of the other hexokinase reactions, so that in glycogenesis from fructose or galactose, insulin influence does not exist. Glycogenesis from these hexoses can take place in the liver in the absence of insulin.

Figure II gives us information about the structure of the glycogen molecule. Glycogen is made up of a series of glucose molecules. In its straight chain there are linkages between the number one carbon of the first glucose and the number four carbon of the second glucose, and so on. This linkage is created with the help of phosphorylase. However, only straight chains are formed in this way, and in order for the normal tree-like structure of glycogen to

develop, branches of the chain have to grow. These branches contain a linkage between carbons one and six respectively of two glucose molecules in the presence of branching enzyme.

Thus, in order for the normal glycogen molecule to be built up, not only the enzyme phosphorylase is needed, which removes phosphate from glucose-1-phosphate and creates the α-1, 4 linkage, but the branching enzyme is needed which creates the α-1, 6 linkages. In the degradation of the glycogen molecule, phosphorylase will strip off glucose molecules down to the branch, but then the debrancher molecule has to split the α-1, 6 linkage at the branch point in order for the phosphorylase to continue its action. Thus, the two enzymes act in concert to induce glycogenolysis.

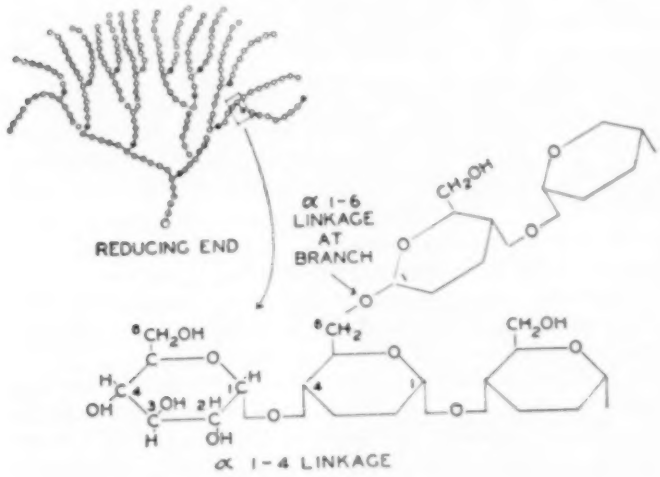

Fig. II. α 1-4 Linkage

Fig. 11 The glycogen molecule as depicted by G. T. Cori. Open circles are alpha 1-4 glucosidic links and solid circules are 1-6 links. Segment at the branch point is enlarged to show structure.

The working out of the structure of the glycogen molecule by the Cori's and their associates is one of the classics of biochemistry.

Figure III summarizes the different types of glycogen storage disease that have been identified so far.

The most common type of glycogen storage disease is known as von Gierke's disease. It comes on in early infancy, and is characterized by retardation of growth, low blood sugar, evidence of excessive metabolism of fat, with ketosis and hyperlipemia, and no rise in

blood sugar after epinephrine, or at most a slight rise. A similar lack of response occurs to glucagon. These children show hepatomegaly and they usually die of infection. Their kidneys may also be enlarged.

The glycogen of these children is found to be of normal structure, but analysis of the enzymes in the liver has disclosed in almost all cases that there is either absence or marked diminution of glucose-6-phosphatase, the enzyme which converts glucose-6-phosphate to glucose and phosphate and permits glucose to get into the blood stream.

The second type of glycogen storage disease is quite rare and affects primarily the heart, but also the tongue and other portions of the body. It is a disease characterized by progressive and intractable heart failure with cardiac enlargement sometimes to five and six times normal.

On histological examination, there is considerable replacement of myocardium by glycogen. The reason for such accumulation or deposition of glycogen is not known because, to date, no abnormality of the glycogen molecule itself has been identified and no absence of glycogenolytic enzymes has been found.

Figure III

SUMMARY OF GLYCOGEN STORAGE DISEASES

Type	Organs Affected	Age		Hypoglycemia, Ketosis, Hyperlipemia	Epinephrine Response	Glycogen Structure	Enzymatic Alteration
		Onset	Death				
I	Liver; Kidney	Neonatal; Infancy	Varies with severity	+++++	0+++	Normal	Decreased to absent glucose-6-phosphatase
II	Heart muscle, tongue, brain	Neonatal; Infancy	Early	0	Normal	Normal	None found
III	Liver (cirrhosis); reticuloendothelial system	Late Infancy	1-10 yr.	0	++	Abnormal; excessively long outer branches	Probably decreased brancher activity
IV	Liver; muscle	Late Infancy	?	++	+	Abnormal; very short outer branches	Probably decreased debrancher activity

Hers, L. Am. J. Med. 24:54 1958

The third type may be pertinent to the case that was presented today. This is a rather rare form of glycogen storage disease. It is characterized by the presence in the liver of an abnormal form of glycogen which resembles amylopectin, the branch component of starch. There seems to be deficiency of brancher enzyme and the resulting glycogen molecule is not normal, producing a foreign body reaction in the tissues. As the abnormal glycogen accumulates in

the liver, fibrotic reaction occurs around it and in addition to accumulation of glycogen, cirrhosis of the liver may occur. At the same time there is no remarkable absence of available sugar. There is a diminished output of glucose by the liver, the epinephrine response is reduced but not absent, and the symptoms are primarily those of modified glycogen storage disease with superimposed cirrhosis. Depending on the severity of the disease, death may occur in infancy or after ten years or more.

The fourth type of glycogen storage disease is a variant of the first type, and only two or perhaps three, cases of this type have been reported. Instead of an absence of glucose-6-phosphatase, there is a lack of debrancher enzyme. As far as I know, this particular type of glycogen storage does not produce the same degree of cirrhosis of the liver as described in type III. It is merely another form of what we think of as von Gierke's disease.

I might say a few words about diagnostic approaches to these disorders. The ideal diagnostic test for glycogen storage disease might be to take some of the liver material and, after showing by suitable staining techniques and analyses that it contains an excessive concentration of glycogen (which is difficult on small bits of liver), to apply the techniques developed by the Cori's and their associates to identify the glycogen as normal or abnormal. This is a rather complicated procedure. Then, one can determine whether the hepatic glucose-6-phosphatase is present, absent, or reduced.

A simpler and more practical approach is to administer a substance that stimulates liver glycogenolysis. If something is wrong with the glucose-6-phosphatase mechanism, the blood sugar will rise only slightly or not at all after a dose of epinephrine or glucagon. The only situations in which a patient might fail to show a hyperglycemic response to these agents are (1) glycogen storage disease, (2) glycogen depletion secondary to drastic restriction of the diet, (3) severe parenchymal liver disease, and (4) severe diabetes mellitus.

The patient that was presented today may have had enough liver disease *per se* to account for the diminished response to glucagon and epinephrine. It has been shown that in liver disease the hyperglycemic response to glucagon and epinephrine is reduced.

With the qualifications just noted, the hyperglycemic response to glucagon represents a good diagnostic test for glycogen storage disease. There is another refinement of this test which I have tried only once. If glucagon is administered, (glucagon and epinephrine both influence the phosphorylase reaction), the amount of active

phosphyorylase in the liver is increased and the rate of transformation of glycogen to glucose-1-phosphate is accelerated. The glucose-1-phosphate that is formed has to go somewhere, and one might reason that it would travel down the Emden-Meyerhof pathway to become pyruvate and lactate. It is my feeling, supported by one experience, that in the patients with glucose-6-phosphatase deficiency, glucagon administration will increase pyruvate and lactate formation. In this case liver glycogenolysis accompanied by a rise in lactate or pyruvate in the peripheral blood, while a rise in blood glucose does not occur. Such a response would seem to localize the defect at the glucose-6-phosphatase level.

If, on the other hand, a deficiency of debrancher enzyme is present, or there is an abnormal glycogen molecule, glucagon administration will fail to induce a rise in blood pyruvate or lactate and the blood glucose also will not show any appreciable change.

DR. EAGLE: Dr. Hofman has had some experience with liver disease in the neonatal period which may explain this patient's condition.

DR. ADELE HOFMAN: Ass't. Attending Pediatrician. I would like to make two comments. First the history of jaundice in the neonatal period is not characteristic of glycogen disease, but is of other forms of juvenile liver disease. I do not think we are able to differentiate between cirrhosis following an early hepatitis and the juvenile types of cirrhosis which start principally with fibrosis and not too much cellular disease.

Several years ago Dr. Ruth Harris of Babies Hospital and I became interested in the derangement of glycogen metabolism in children with liver disease other than glycogen storage disease. Such a derangement is extremely unusual although glucose-6-phosphatase activity is markedly reduced in biopsy and autopsy specimens from all forms of liver disease. There have been a number of reports in adults where hypoglycemia has complicated carcinomatosis and also cardiac failure.

In the literature we found only six cases of hepatogenous hypoglycemia in children which were not due to glycogen storage disease. One of these was secondary to phosphorus poisoning; one with idiopathic, acute, yellow atrophy; three with undiagnosed types of hepatitis; and one child who had jaundice in infancy developed hypoglycemia two years later.

At Babies Hospital we found six more children who had manifestations of hypoglycemia with liver disease. One of these had

acute yellow atrophy following vinethene anesthesia for tonsillectomy. One child originally diagnosed as glycogen storage disease because of hepatomegaly and two episodes of coma subsequently turned out to have a hepatoma. The third was an infant with idiopathic hepatitis who presented first with hypoglycemia and later developed jaundice. The last three infants of our group had had hepatitis in infancy and from six months to two years after complete disappearance of all liver abnormalities developed hypoglycemia.

The child presented today adds a 13th case to the literature and our Babies Hospital series. He apears to have definite derangement of his carbohydrate mechanisms without other liver dysfunction.

Dr. Eagle: In summary, we have presented a patient who responds to epinephrine and glucagon administration in a fashion consistent with a diagnosis of glycogen storage disease. However, the history of neonatal jaundice suggests that he may be actually suffering from an even rarer condition, namely a disturbance in carbohydrate metabolism secondary to liver damage without any other evidence of liver dysfunction.

(Subsequent to this conference, the liver was analyzed by Dr. Joseph Sokal and Dr. Edward Sarcione, Roswell Park Memorial Hospital, Buffalo, New York. The glycogen content was 1.8%, slightly above normal but well below that found in conventional von Gierke's disease where it varies between 7-15%. Upon incubation the glycogen fell from 1.8% to 0.8%. When fresh rat liver was added to an hemogenate of the liver specimen, all the glycogen disappeared. This suggests that the molecular structure of the glycogen was normal. Because the liver became thawed in transit to Buffalo, glucose-6 phosphatase determinations, although low, were not considered accurate.)

The April 1934 issue of *Archives of Pediatrics* presented to its readers this original and excellent fifty-year review of pediatric progress by Dr. Louis Fischer. Now marking the completion of its 75 years in the field of pediatrics, this journal re-publishes the late Dr. Fischer's noteworthy paper.

To give full and proper significance to the extraordinary forward strides made during the *past quarter-century* in this medical specialty, *Archives* will—in January 1959—present a highly informative report, "Pediatric Progress During the Last Twenty-Five Years," by Murray H. Bass, M. D., Consulting Pediatrician, Mt. Sinai Hospital, N. Y., and late Associate Clinical Professor of Pediatrics, Columbia University.

PEDIATRIC PROGRESS DURING THE LAST FIFTY YEARS*

1884 - 1934

By Louis Fischer, M. D.

New York

What progress has pediatrics made during the last fifty years? In 1883 to 1884 there were two schools of thought which predominated: one, the School of Pathology by Virchow; and two, the New School of Bacteriology. Pasteur had worked bacteriologically in preventing the ravages of the silk worm a number of years before Koch. Pasteur also had demonstrated that fermentation in wine and in milk was caused by bacteria. The famous lactic acid milk, according to Metchnikoff, originated in the Pasteur Institute in Paris. Pathologists did not recognize pathogenic bacteria as the cause of infections. They sneered at the presence of a pneumococcus or a streptococcus in sputum and did not believe that the Klebs-Löffler bacillus could cause diphtheria. Klebs reported his work in 1883, and Loöffler published his report independently in Berlin in 1884. The staphylococcus and streptococcus had been described by Pasteur in 1878 and 1879. In 1883 Fehleisen described bacteriologically the streptococcus causing erysipelas. The tetanus bacillus was described by Nicolaier in 1884. Thus fifty years ago was an epoch making series of discoveries which have laid the foundation for bacteriology. Nor must I forget Koch and his discovery of the tubercle bacillus in 1882. About this time also Eberth described the typhoid bacillus.

* Read by invitation before the Clinical Society of the Elizabeth General Hospital, Elizabeth, N. J., February 20, 1934.

Let us consider one disease—diphtheria. The treatment consisted in giving bichloride of mercury with or without iron in doses of 1/30th increased to 1/20th and 1/10th of a grain in young children every few hours. It was surprising to note how septic diphtheria could tolerate enormous doses of bichloride of mercury. Laryngeal stenosis caused by croup was so common that it was not unusual for me to see every week in the homes of private patients several cases requiring intubation. Older physicians present will agree with me that 20 to 25 years ago they saw fifty and many more cases of diphtheria in the course of a year in their private practice. Very many of these had laryngeal stenosis and croup. The croup kettle and the use of ipecac, with and without squills, was an every day remedy. How different today! We must give massive immunization the credit for having prevented thousands of cases of diphtheria. If you will study the statistics of positive and negative Schick reactions after immunization with toxin-antitoxin or toxoid, you will find as many as 1/5th of all cases of toxin-antitoxin immunization in which one could not produce immunity. Toxoid, however, seems to offer better results. It is not in use long enough to make positive statements in comparison with toxin-antitoxin, which is practically abolished by the New York Health Department. Forty years ago O'Dwyer published his famous paper on intubation in laryngeal stenosis. This superseded tracheotomy which was in vogue at that time. Do not imagine that laryngeal stenosis in diptheria was our worst complication. Laryngeal stenosis accompanying measles usually ended fatally, despite antitoxin and despite careful intubation. When antitoxin was introduced in 1895, there was considerable opposition to its use. I remember reading a paper at the Post-Graduate Medical School in 1894 and also at a clinical meeting at the New York Polyclinic in 1895. One of the gentlemen invited to discuss my paper said he would no more use antitoxin in the treatment of diphtheria than he would inject his cases with horse's urine.

Scarlet fever was the subject of an intense study in Vienna by Moser and Escherich about 1890. They found a streptococcus always present in cultures. Marmorek developed an antistreptococcic serum which was supposed to be a specific in scarlet fever as antitoxin is in diptheria. This serum was a failure. Mallory and Dick made extensive studies resulting in the Dick test so well known today. American studies have shown that there are very many

varieties of streptococcus hemylyticus but this latter microorganism is most likely the specific factor causing scarlet fever. The Dochez strain, as well as the strain number 2, is usually found in scarlet fever.

Although beriberi, in 1879, was the cause of 32.8 per cent. of deaths in 100 patients on a diet of rice and rice only and 10 years later, in 1889, on a diet of meat, vegetables, barley, etc., we find that beriberi amongst the Japanese had almost disappeared. In other words, by eliminating polished rice from the diet and giving unpolished rice, there was but one case of beriberi amongst 10,000 Japanese prisoners. Funk was able to extract the vitamin in 1911 which was the antiberiberi element present in unpolished rice.

Infant feeding is a simple problem in the normal child. It is complicated when we are dealing with a premature or with a dyspeptic or atrophic child. Such children are difficult to feed and a safe rule is to begin with skimmed milk and carbohydrate. The question of carbohydrate is one of individual tolerance. Personally, I like Dextri-Maltose. I also favor sucrose 5 per cent. or more, up to 15 per cent. at each feeding. On the other hand, where there is a tendency to constipation, lactose should be given. Beta Lactose is a very satisfactory preparation and can be given in 5 to 10 per cent. of the gross amount of food, or one ounce of lactose to 20 ounces of milk.

Have I forgotten mother's milk? Not at all. This is our standard food and should always be utilized first. When mother's milk is deficient, one can always give a supplementary feeding of cow's milk. A word concerning dry milks. Klim and Dryco, as well as condensed or evaporated milks, all have their places as a substitute in infant feeding or as supplementary feedings. Especially in travelling and during the night, when an emergency may require a substitute feeding, Dryco or evaporated milk is indicated. Where intestinal indigestion with stools containing saponified fats or an excess of protein curds are noted with stationary weight, let me advise the use of casein lactate in the form of lactic acid milk. To those of you who know Marriott's method, follow it, using Karo corn syrup as a carbohydrate. My experience with the same in atrophic infants and in those who do not seem to tolerate the ordinary casein is so good that I would strongly advise you to try this when there is an opportunity. Even when human milk disagreed, and I have seen such cases, the casein lactate milk plus Karo corn syrup seemed advantageous.

May I dwell for a moment on feeding in pyloric spasm. You have all seen cases in which vomiting of food was continued after each milk feeding. Water and all iquids were ejected, likewise broth and soup. These cases of pyloric irritation do very well when fed thickened farina. The farina can be cooked in milk and sugar added. Sugar renders it more palatable, besides increasing its caloric value. In cases where emaciation has followed this continuous vomiting and where the Rammstedt operation was considered as a last resort, thickened cereal feeding gave me good results. An occasional case will not respond to this treatment, but in very many cases excellent results have followed the use of thickened pap. Cereals, such as farina, rice, and barley, are prescribed by many pediatrists as routine food in early infancy. Other pediatrists prefer vegetable purees and vegetable juices during infancy. Pap made of bread and milk, or zweiback and milk was advised several hundred years ago. The oldest book available in recent years is by Vallembert, 1565. Ettmuller, in 1698, also advised some form of pap during infancy. Buchan, in 1776, advised giving once or twice a day either a weak broth with bread in it or a cereal after the third or fourth month. In 1794 Morse advised the addition of bread to the infant's diet after the end of the second month. In 1754 Brouzet advised the use of pap made with beer rather than pap made of cereal or bread with cow's or goat's milk. Brouzet insists that during the weaning period bread and milk are necessary. A book published by Rosenstein in 1776 is interesting because he opposes the use of cereal. He says verbatim "that it is gluey, tenacious, and a slimy food causing obstruction in the mesentery, hardness in the stomach, acidity and win by which we lose at least one-half of our children". DeClaubry, in 1783, was a strong opponent to the use of cereal. Thus we can find opponents and strong advocates of cereal feedings during infancy.

Fads predominate in infant feeding. It is not more than 30 or 40 years ago that high fat feedings were advised. Not only did we see infants receiving 3, 4, 5 and 6 per cent. feedings ordered as routine, but the main point was to show large gains in weight. Later on this was superseded by high protein feedings, and more recently high carbohydrates are very much more in vogue. You will also remember how Soxhlet advised the sterilization of milk and how later on pasteurization was substituted. Pasteurization, heating milk for 20 minutes to 145 degrees, really accomplishes all one desires, for it destroys pathogenic bacteria of of which the tubercle bacillus is the most dangerous. Boiling the milk is the simplest and safest method.

Between 1895 and 1900 Rotch, then Professor of Pediatrics at Harvard University, read a paper before the American Pediatric Society advocating percentage feeding. His idea was that the physician should prescribe so much fat, so much sugar, and so much proteid just as the doctor would order drugs. Order a series of bottles for 2-hour, 3-hour, or 4-hour interval of feedings. Thus, from six to ten bottles to be used in 24 hours were delivered every morning and the empty bottles were called for the following day.

The Walker Gordon Laboratory opened a laboratory in most of the larger cities of the United States and this method of percentage feeding was followed all over the country. It did not take long to note that the infants did not do well on this method of feeding and so the method was changed from time to time. A new-born baby, for instance, was given a formula of 2 per cent. fat, 5 to 6 per cent. carbohydrate, and 1 to 2 per cent. proteid. This was made up with barley gruel and looked very attractive. It was, however, quite expensive, which was a drawback to its popularity.

A great step forward was made during the last quarter of a century by the discovery of the causes of deficiency diseases. These deficiency diseases are frequently described as cases of avitaminosis. By this is meant diseases which originate from a deficiency in certain vitamins. Funk was a pioneer in this research work, followed by Hess, McCollum, Sherman, Steenbock and many others.

The vitamins can be taken care of by giving fruit and vegetable juices. The leafy vegetable juices, expressed from steamed spinach, lettuce, carrots, broccoli, brussels sprouts or sauerkraut, are well adapted for the infant and can be given as early as two to three months. They contain the earthy salts and vitamins so necessary at this early age. As a rule, I give these vegetable juices *like water between meals.* In many instances infants will have loose bowels from spinach juice, which must then be omitted. To supply vitamins A and D in the deficiency diseases, cod liver oil, halibut liver oil and salmon liver oil must not be omitted. These may be given with or without orange juice after the infant is one or two months old. Where fat intolerance exists, vomiting may follow the dose of cod liver oil. As an antirachitic factor, sunlight is very important. When sunlight is not available, the ultraviolet ray can be used as a substitute. One should not forget that calcium deficiency in infants can be made up by giving cod liver oil. Viosterol has many adherents, but it also has many opponents. In a recent publication the following statement is made: "Viosterol is not the equivalent of cod liver

oil." Barnes reports that a group of infants receiving 1250 units of vitamin D in the form of viosterol were no more protected against rickets than an untreated control group. He further reports that in another series of infants receiving about 1,400 units of vitamin D in the form of cod liver oil, 95 per cent. were protected. De Sanctis and Craig published a similar study. Hess, in a recent publication, states that he has been disappointed in viosterol. The consensus of opinion is that viosterol does not afford the same protection as that obtained with cod liver oil. Under such circumstances one should be careful not to pin his faith to the specific virtues of viosterol until all clinicians agree that it is specific. Hess states that incomplete protection was afforded unless we give the equivalent of 10 teaspoons of cod liver oil daily; in other words, the presupposed equivalent of 20 drops of viosterol.

The credit for having described lumbar puncture, about 1895, belongs to Quincke. Needless to say, in cerebrospinal meningitis, tuberculous meningitis or in poliomyelitis, it is a most valuable diagnostic aid in differentiating these conditions. In some forms of encephalitis, with thickening and suppuration at the floor of the 4th ventricle, we may have a dry tap. In such cases if a puncture between the 3rd and 4th, or 4th and 5th lumbar vertebra yields no fluid, then cistern puncture can be tried. One should always disregard the age of an infant and be on the alert for acute meningeal infections as the following case shows.

An infant, 7 weeks old, was admitted to my service at the Sydenham Hospital many years ago. It had a marked rigidity of the sternocleido mastoids, opisthotonos, vomiting and fever. There was marked bulging of the fontanel. Lumbar puncture resulted in a dry tap. After several dry taps were noted, we aspirated the lateral ventricles. Several ounces of a thick purulent fluid were aspirated. This contained the diplococcus intracellularis. Not having had any experience with this type of cerebrospinal meningitis, I invited Dr. Simon Flexner to see this child. He advised me to aspirate both lateral ventricles, then wash the ventricles with a normal saline solution until the fluid returned clear, and then replace the aspirated fluid with 30 to 50 c.c. of antimeningitic serum. This procedure was used daily. He also sent Dr. Jobling to further advise me. The case was in the hospital about 5 weeks and made an unenventful recovery. I presented this case before the Section of Pediatrics at the New York Academy of Medicine in 1910. This case was followed up by me for a number of years until the child went to school. It was a perfectly normal child, mentally and physically.

The era of bacteriology developed the use of sera, vaccines and

autogenous vaccines. While pertussis serum is occasionally beneficial as a prophylactic, it has not shown any specific therapeutic virtues.

Mass immunization is the great problem of the future. This is being tried in measles where convalescent serum in doses of about 5 c.c. or more can prevent a measles infection.

In 1885 Pasteur described a case of an Alsation boy in whom rabies was prevented and cured by an inoculation with antirabies vaccine.

One of the greatest advances made during the last half century is the prevention of blindness in infancy. Consider that there were 6,200 cases of blindness reported in the State of New York in 1906. Of these, most of them could be attributed to opthalmia neonatorum. A careful study showed that 1,984 were preventable by the instillation of one drop of 1 per cent. nitrate of silver solution on the center of the cornea. More recently, a 10 per cent. protargol solution has been substituted for nitrate of silver. It is just as effective and less irritating. The gonococcus is so frequently found in the vaginal secretions of women delivered in lying-in asylums that the wisdom of prophylaxis cannot be questioned.

Placental extract in doses of 2 to 5 c.c. is now being used to convey immunity. In women who have been Wassermannized, this prophylaxis is being tried in some instances to protect children exposed to various infections. Whether or no mass immunity can be effected in this way to protect a community against poliomyelitis is an important thought.

The following interesting tests have lived through considerable opposition. They are the Mantoux test, the Pirquet test, the Schick test and more recently, the Schultz-Charlton test (blanching) in scarlet fever. An intense study of the Calmette vaccination as a prophylactic against tuberculosis in early infancy has been made by Pirquet and Nobel in Vienna. Both of these gentlemen informed me that they were opposed to this method as it does not protect as suggested by Calmette. Finkelstein of Berlin and Schlossmann of Dusseldorf, with Eckstein, not only used the Calmette vaccination in infancy but also used it in a large series of monkeys adapted for this test. Last summer, at the Medical Congress in London, Eckstein told me that he discarded the use of this prophylactic measure entirely. Eckstein and his predecessor, the late Prof. Schlossmann, devoted several years to this study which they published elsewhere.

In 1901 I was appointed an attending physician to the North Brothers Island Hospital. On my service at the smallpox wards

I found severe cases of smallpox treated by allowing sunrays or light to filter through red glass windows. It was claimed for this treatment that the pitting in smallpox is much less. This method of treatment has been abandoned.

One word concerning the announcement of x-ray by Roentgen in 1895. No greater aid has ever been given to the profession than the use of x-ray. The x-rays should be used and carefully interpreted only as an aid to the symptoms which one notes by clinical observation. The interpretation of x-rays in rickets, in syphilis affecting the bones, and in scurvy needs no comment from me but it is in the interpretation of intracranial lesions, such as neoplasms, that one should be careful. I would not advise an operation for a mastoiditis based solely on an x-ray. On the other hand, let me point out but one instance in which an infant or young child will be brought for an opinion. Its appetite is poor. It does not seem to gain in weight. It is restless. It has but a slight rise of temperature in the evening. It is not a happy, healthy child. In such cases one will frequently find an early manifestation of pulmonary tuberculosis by means of the x-ray and this long before cough or expectoration or fever is present, and before cavities exist.

Let me point out a second instance in which fever is present and in which there may be vomiting or gastric disturbance with abdominal pain and no physical signs in the chest. In such cases a consolidation of the lung or a central pneumanic process is the cause of our trouble. Here the x-ray renders marvelous aid. Obscure lesions, in which choking of the disc may presuppose the existence of a cerebellar tumor, will be aided by an x-ray of the skull. When symptoms of an acute appendicitis are present, and one has made a differential leukocyte count, then a clinical diagnosis is ample. The operation should not be delayed for an x-ray picture, for an acute condition may result in a ruptured appendix with its associated dangers. An x-ray in such cases is superfluous.

We are indebted to Banting for his contribution to the therapy of infantile, juvenile and adult diabetes with the therapeutic aid of insulin. This brings me to the subject of endocrine therapy. Cretinism and its treatment with thyroid is perhaps a specific instance in which positive results can be foretold. On the other hand, many of our endocrine secretions have disappointed me as they will you in many cases in which they are recommended. The virtues of the pituitary gland during infancy and ovarian extract are not as specific in their results as they are claimed to be. One must, however, laud the virtues of the adrenal glands. Adrenalin, epinephrine, neosynephrine and epinine all have remarkable virtues in the manage-

ment of conditions in which a vasoconstrictor is indicated. The virtues of pituitrin in obstetrics you will understand are not under discussion tonight. My point is to review those endocrine secretions which need emphasis in pediatrics.

Pediatrics has made great strides in the use of anesthetics. One cannot forget the crude method of giving chloroform and also ether for short or long operations. Pulmonary and kidney complications have frequently followed prolonged ether anesthesias. During recent years, gas and ether, or ethyl chloride and ether for short anesthesias, seemed to give the best results. Local anesthesias can be used in brain surgery or in abdominal surgery. I believe with the use of avertin in skillful hands and without fear of complications that we have a drug that will probably displace the former anesthetics, first, because it acts so quickly; and second, because of no after effects.

One word concerning transfusion. This is a most valuable remedy in infancy regardless of age. In the premature infant one ounce or more of blood, injected intramuscularly aided by human milk, will frequently work well. The use of blood transfusion in hemophelia must not be forgotten. We do not fear the danger of hemolysis as one does in the transfusion of an adult. The following is an interesting case of a convalescent scarlet at the Willard Parker Hospital. The child was septic, had been extremely ill for one-half dozen or more weeks, and had had every complication imaginable, including kidneys and ears. It did not respond to restoratives, including iron and arsenic. A transfusion was suggested. We gave the child 250 c.c. of citrated blood with no evil effects. On the day following the injection, the child had a chill and temperature. This recurred every other day. The blood examination showed plasmodia.

Allergy and allergic conditions, such as asthma, eczema, protein sensitization and serum sickness, are mentioned as another important progressive step. The value of food tests and hay fever tests need no emphasis by me.

What has been said concerning mass immunization for the elimination of diptheria as a prevalent disease applies even more strongly to the benefits derived through the inspection and hygiene of the cows. When it was found that milk brought to this city frequently contained millions upon millions of bacteria the Health Department graded the milk so that one knew which was excellent milk and which was good milk. They graded it according to grade A, grade B and grade C. Every summer had its thousands of cases

of cholera infantum, otherwise known as enterocolitis, caused by infected milk. All the large cities had the same disease in summer, and the mortality was exceedingly high. In this country, thanks to the efficiency of the Health Departments in securing clean milk, this disease is practically wiped out since the last 15 to 20 years, but enterocolitis was by no means confined to American cities. We saw the same diseases in Berlin, Paris, London and Vienna at all the clinics.

Not more than 25 years ago it was customary to do a tonsillotomy. This consisted in pulling the tonsil forward and using a guillotine, cutting off the large piece of tissue. This has all been discarded and only tonsillectomy used. Tonsillotomy was a primitive operation compared to the thorough method of today.

I have tried to give some of the highlights of what has transpired in pediatrics, but naturally in the time allotted for this paper one cannot give a complete résumé of many therapeutic and diagnostic advances which will aid in the diagnosis and therapeutics of pediatrics. I therefore crave your indulgence if I have omitted points which to you seem important but which have not been mentioned this evening. I thank you for the privilege of appearing before you and presenting my remarks as a modest report of the last half century of pediatric progress.

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Current Literature

Edited by

MICHAEL A. BRESCIA, M.D.

WIDELL, S.: ON THE CEREBROSPINAL FLUID IN NORMAL CHILDREN AND IN PATIENTS WITH ACUTE ABACTERIAL MENINGOENCEPHALITIS. (Acta Paediatrica 47: Suppl. 115, May 1958).

In an investigation of the cell content, total protein content and the electrophoretic protein pattern of 123 cerebrospinal fluid samples from 98 normal children, aged 0-13 years, the following observations were made.

The cell count of the CSF was higher during the first 3 months of life than later in childhood. The range of variation of the individual values was considerable, above all during the first weeks of life.

The total protein content of the CSF was also larger during the neonatal period than during the rest of childhood. During the first year of life the mean protein content decreased successively, reached a minimum between the ages of 9 months and 2 years, then began to increase again and continued to do so throughout the rest of childhood.

Of the electrophoretically separated protein fractions of the CSF, albumin in its variation with age showed a distinct parallelism with the total protein content. The mean albumin concentration in the CSF was thus highest during the neonatal period and lowest in children 9 months to 2 years of age, after which it again successively increased. The other protein tractions of the CSF, except gamma-globulin, showed roughly the converse variation.

During the first week of life the relative concentration of albumin in the CSF was higher than in the serum. The physiologic basis of this is presumably mechanical irritation of the meninges in association with delivery and subsequent increase of passage of serum protein into the CSF space, especially of the relatively small dispersed albumin molecules.

The variations in the amount and composition of the CSF protein in different ages probably depend on variations in the relative amount of the admixture of serum protein during passage of the CSF from the ventricles to the lumbar subarachnoidal space. The increased addition of serum below 9 months of age is probably due

to an increased permeability of the blood-CSF barrier. The changes occurring after 2 years are probably related to a relatively decreasing function of the choroidal plexus with increasing age.

Analyses of 256 CSF samples from 119 patients, including 60 children, with acute abacterial meningo-encephalitis revealed the following.

At the time of onset of the disease the cell content of the CSF was usually higher in mumps meningo-encephalitis than in meningo-encephalitis of other origin. While no relationship was found between the CSF changes and the clinical picture during the acute stages of mumps meningo-encephalitis, a clear correlation was demonstrable in meningo-encephalitis of other origin between the clinical symptoms at onset and the cell and protein content of the CSF.

During the later course of the disease most patients with mumps meningo-encephalitis were symptom free. In meningo-encephalitis not related to mumps, however, persistent symptoms in the form of tiredness, headache, dizziness, loss of memory, concentration difficulties, irritability, abnormal intolerance of light and sound were more frequent and to a large extent accompanied by pronounced changes in the CSF, i.e. marked increase in the cell content, protein content and the relative concentration of gamma-globulin. It is difficult to judge to what extent the brain was involved by the pathologic process. Symptoms of encephalitis in the form of persistent pareses or tendency to convulsions were noted in only 2 cases. It is true that the symptoms dominating the picture during the later course suggested cerebral involvment but they may also have been partly of meningeal origin.

Of the electrophoretically separated protein fractions of the CSF, the albumin during the initial stage of the disease was markedly increased, the relative concentration exceeding that in serum, at the same time as the other protein fractions of the CSF were below normal. During the further course the pattern gradually became normal although the relative gamma-globulin concentration was above normal for some time afterwards, especially in patients with severe symptoms.

Judging from the results obtained, the changes in the CNS in mumps meningo-encephalitis are probably relatively benign and localized mainly to the meninges. The other meningo-encephalitides represented a heterogenous group with respect to the etiology, severity and duration of the disease and probably also regarding its pathologic-anatomic localization.

The abnormalities of the electrophoretic pattern of the CSF protein are probably due to disturbed meningeal permeability.

Electro-encephalography was performed in 58 cases (13 with mumps meningo-encephalitis and 45 with meningo-encephalitis of other origin). Of the recordings, 17 showed slight abnormalities in the form of mild theta dysrhythmia, while 9 showed more pronounced changes with delta focus. The remaining 32 recordings were of normal appearance. The CSF findings of those with abnormal electro-encephalograms did not differ from those with normal EEGs. M. A. B.

BOOK REVIEWS

EVERYDAY PROBLEMS OF BOYS AND GIRLS. By SIDONIE MATSNER GRUENBERG. Cloth. Pp. 363. Price $4.95. New York, Random House, 1958.

Sidonie Matsner Gruenberg, as author, editor, lecturer and special consultant to the Child Study Association of America, is well known for her contributions to the field of welfare and guidance of children. In her most recent book, she has written a guide or manual for parents in meeting and handling the everyday problems of their children. Books, pamphlets and articles dealing with this subject are indeed abundant, and by no means unique. However, in this latest work, Mrs. Gruenberg offers guidance to parents of children of the "neglected age," namely, five to twelve—or school age children. Mothers have received information from pediatricians, clinics and child care centers about the proper care of infants. Likewise, as the author reminds us, the teen-agers and adolescents have been receiving a great deal of attention. However, we are reminded that there has been too little in the literature about guidance and understandnig of children of the middle years. During this age, boys and girls, although less demanding than the age that precedes or follows them, must cope with their own unique problems of growing up. These problems involve friends, family, school and community. The author draws upon the experience of actual cases; real parents and real children, with real problems, living in both rural and urban communities. Parents are often aware of the differences between boy and girl, but in this book they are reminded of the individual differences between girl and girl, and boy and boy. Apart from differences in needs, learning and growth, the author calls attention to the uneveness of maturing, and in general, the unpredictability of these years. Besides growing fast, the child from five to twelve is

also wondering, experimenting and, of course, playing. The first part of the book, which is entitled, "At School—At Home—At Play," deals with the activities of the child's daily life. The second part, "Health and Growth," is an excellent manual, and should interest pediatricians as recommended reading for parents. Outstanding among the chapters of Part II, is one dealing with the handicapped child. It is realistic, direct, and approaches the problem by means of sound psychological principles. Part III, "Character and Responsibility," discusses the subjects of responsibility, sex and money. An excellent, but not exhaustive, reading list is appended. This reviewer feels that an important and definitive work should have been included among the titles, namely, Arnold Gesell's, "The Child from Five to Ten." The latter is still an outstanding book and a deservingly popular guide in the ten major fields of behavior.

ELEANOR ROTHENBERG, M.A.

PARENT-CHILD TENSIONS. By BERTHOLD ERIC SCHWARZ, M.D. AND BARTHOLOMEW A. RUGGIERI, M.D. *Cloth*. Pp. 238. Price $4.95. Philadelphia, Pa.: J. B. Lippincott Company, 1958.

The authors were quite successful in explaining in nontechnical language and with a minimum of psychiatric jargon the behavior problems of children and how they are produced or influenced by the emotional background of the parents. The authors address themselves particularly to the parent. However, this reviewer questions the validity of this approach. The parent who needs the type of advice that is contained therein could no more benefit by reading the text than a patient with acute appendicitis could be benefited by reading how to do an appendectomy. There is also a tendency to oversimplification which might be misleading to the nondiscriminating reader. For example, the chapter headed Emotional Aspects of Feeble-mindedness is an isolated example of a cause of feeble-mindedness which was helped by understanding the underlying emotional tensions of the parents vis-a-vis the child. However, this was actually a case of pseudoretardation and the nonmedical reader might get the impression that most or all cases of feeble-mindedness are due to emotional disturbances which of course is palpably not so. The authors in their next edition might very well address themselves to the medical profession. This can be done very easily by slight changes in wording and some modifications in the text. The book would then be of great value to the physician who is not too well versed in this particular field.

M. A. B.

AUTHORS' SUMMARIES

EMMONS, C. W.: ASSOCIATION OF BATS WITH HISTOPLASMOSIS. (Public Health Reports 73:590 July 1958).

Histoplasma capsulatum was isolated from 50 of 105 soil samples from 6 collections made between October 1596 and September 1957 on the premises where a family epidemic of histoplasmosis had occurred. Chickens, often associated with the saprophytic growth of this fungus, had not been kept on the premises for many years.

Histoplasma was isolated from 45 of 66 soil specimens taken adjacent to or within 5 feet of the foundation wall of the house and from only 2 of 29 taken 6-18 feet from the house. Three of 10 samples taken near a doghouse were also positive.

The house sheltered a colony of the brown, or house, bat (Eptesicus fuscus), and bat dung was found adjacent to the foundation. The presence of the bats is the apparent factor responsible for the constant saprophytic infestation of soil on these premises with H. capsulatum.

The house bat inhabits suitable shelters in towns and cities, as well as rural areas, and may be the ecologic factor responsible for the presence of Histoplasma, prevalent histoplasmin sensitivity, and clinical histoplasmosis in observed instances of urban histoplasmosis in towns and cities where reasons for the occurrence of these phenomena have not yet been determined.

ZIMMERMAN, M.C.: PENICILLINACE-PROVED ALLERGY TO PENICILLIN IN POLIOMYELITIS VACCINE. (Journal Americal Medical Association 167:1807, August 9, 1958).

Penicillin is present in poliomyelitis vaccine (and adenovirus vaccine) in concentrations varying from 1 to 200 units/c.c., except in Wyeth poliomyelitis vaccine, which contains less than 0.001 U./c.c. There was sufficient penicillin in the vaccine (except in one brand as reported in this paper) to cause allergic reactions in exceptional patients highly sensitive to penicillin. Six such allergic reactions, each duplicating a previous reaction known to be due to penicillin allergy, have been studied. The rapid clearing after one penicillinace injection and the lack of reaction to a brand of penicillin-free vaccine is offered as proof that penicillin, rather than some other allergen, was the cause of the reaction in each patient. In patients with a history of penicillin allergy, penicillin-free vaccines should be used. If this is impossible, penicillinace should be given prophylactically preceding the vaccine injection.

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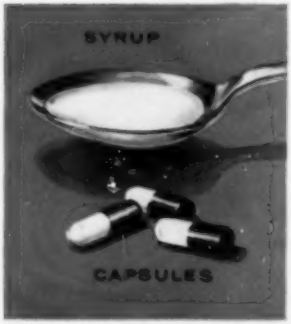

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